without a change in diameter at day 1; however, shear stress was normalized by day 7. Patency was 100% and survival was diminished by 50% in these large AVFs. Shear stress in AVFs created with the 28-gauge needle was decreased by 39% but AVF patency was reduced 50%. Survival was 100%.

Conclusions: This murine aortocaval model faithfully recapitulates the maturation and failure phases of the human AVF; this is the first animal model to show these distinct phases. Shear stress varies according to the diameter of the fistula. and AVF patency depends on the AVF shear stress.

Author Disclosures: R. Assi: Nothing to disclose; **H. Bai**: Nothing to disclose; **K. Brownson**: Nothing to disclose; **A. Dardik**: Nothing to disclose; **M. R. Hall**: Nothing to disclose; **G. Kuwahara**: Nothing to disclose; **J. A. Madri**: Nothing to disclose; **C. D. Protack**: Nothing to disclose; **M. Tsuneki**: Nothing to disclose; **K. Yamamoto**: Nothing to disclose.

PS226.

Significant Increase of Urine Fibrinogen in Mouse Model of Contrast-Induced Nephropathy

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Objectives: Contrast-induced nephropathy (CIN) is the third leading cause of hospital-acquired renal failure. Early diagnosis and treatment of CIN may prevent significant sequelae. It has been reported that urinary fibrinogen (Fg) can be used as a biomarker of ischemia-reperfusion injury in kidney. The objective of this study was to investigate whether urinary Fg can serve as a biomarker for the diagnosis of CIN.

Methods: C57B1/6J mice received a prostaglandin synthesis inhibitor (indomethacin, 10 mg/kg) and a nitric oxide synthase inhibitor (Nω-Nitro-L-arginine methyl ester, 10 mg/kg) intraperitoneally (i.p.), before being given iodixanol in low-dose (6.24 g iodine/mL i.p.) and high-dose (12.48 g iodine/kg i.p.) groups. Mice in the control group received normal saline instead of iodixanol. Urine samples were collected for Fg and creatinine (Cr) analysis using an enzyme-linked immunosorbent assay kit. Kidneys were harvested 24 hours later. RNA was extracted from half of a kidney specimen. Quantitative RT-PCR was used to quantify Fg-α RNA expression, with glyceraldehyde-3-phosphate dehydrogenase as the endogenous control. The other half of the kidney specimen was fixed with formalin, embedded in paraffin, and then stained with hematoxylin and eosin (H&E) for histopathologic evaluation.

Results: H&E stain demonstrated mild renal injury in the low-dose group, scoring from 0 to 1, and moderate renal injury in high-dose group scoring from 1 to 2, based on a pathologic grading scale of 0 to 3. Urinary Fg increased significantly from 0.37 ± 0.13 μg/mg Cr in the control group to 3.46 ± 2.89 μg/mg Cr in the low-dose group ($P < .05$) and 6.15 ± 2.51 μg/mg Cr in the high-dose group ($P < .01$). Fg-α RNA level increased 40% in high-dose group compared with the control ($P < .05$), although no significant changes were detected in the low-dose group.

Conclusions: Urinary Fg level increases are consistent with the pathologic severity of CIN in animal models. These results suggest that urinary Fg may be a potential biomarker for early diagnosis of CIN. Further investigation in clinical patients is needed.

Author Disclosures: H. Dong: Nothing to disclose; **S. Yang**: Nothing to disclose; **L. Yao**: Nothing to disclose; **W. W. Zhang**: Nothing to disclose; **C. Zhao**: Nothing to disclose.

PS228.

Can Edaravone Protect Kidney Damage Caused by Myonephropathic Metabolic Syndrome in Rats?

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Objectives: Free radicals have been implicated in myonephropathic metabolic syndrome (MNMS), which damages not only muscles but also kidneys. At Vascular Annual Meeting (VAM) 2012, we reported the free radical scavenger, edaravone (Radicut, Mitsubishi Tanabe Pharma Co, Japan), suppresses muscle injury by MNMS. In this study, we evaluated whether edaravone can suppress kidney damage by MNMS.

Methods: Lewis male rats (508 ± 31 g, n = 10) were intraperitoneally injected with 3.0 mg/kg of edaravone (edaravone group [n = 4]), or saline (MNMS group [n = 6]). The MNMS models were induced by clamping the bilateral common femoral arteries for 5 hours and then declamping. Five hours after declamping, the both kidneys were stained with hematoxylin and eosin (Fig). Normal kidneys were harvested as a control (n = 3). Kidney damage was evaluated by the number of cells in the glomerulus (glomerulus infiltration) and by the number of residual tubular cells (enlargement of tubular cells).

Results: The number of cells in the glomerulus was significantly increased in the MNMS group compared with the control (77.2 ± 10.3 vs 49.6 ± 2.8 cells/glomerulus, $P < .01$). The number of cells in the

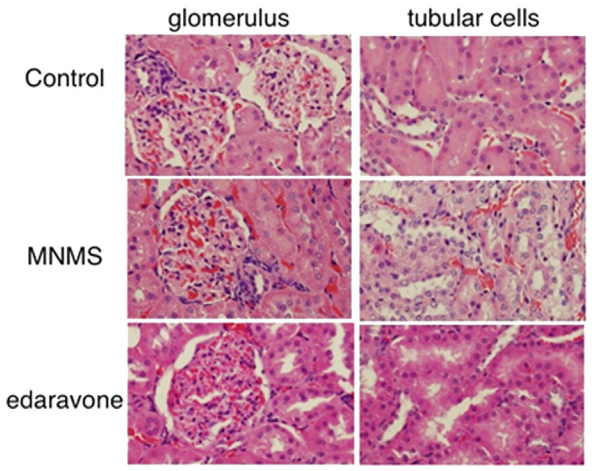

Fig.